## Suppression of proinflammatory cytokines and mediators in LPS-induced RAW 264.7 macrophages by stem extract of Alternanthera sessilis via the inhibition of the NF-κB pathway

## ABSTRACT

Alternanthera sessilis, an edible succulent herb, has been widely used as herbal drug in many regions around the globe. Inflammation is a natural process of the innate immune system, accompanied with the increase in the level of proinflammatory mediators, for example, nitric oxide (NO) and prostaglandin (PGE2); cytokines such as interleukin 6 (IL-6), interleukin 1β (IL-1 $\beta$ ), and tumor necrosis factor alpha (TNF $\alpha$ ); and enzymes including inducible nitric oxide synthase (iNOS) and cyclooxygenase-2 (COX-2) via the activation and nuclear translocation of nuclear factor kappa-light-chain-enhancer of activated B cells (NF-KB) subunit p65 due to the phosphorylation of inhibitory protein,  $I\kappa B\alpha$ . Inflammation over a short period of time is essential for its therapeutic effect. However, prolonged inflammation can be detrimental as it is related to many chronic diseases such as delayed wound healing, cardiovascular disease, arthritis, and autoimmune disorders. Therefore, ways to curb chronic inflammation have been extensively investigated. In line with that, in this present study, we attempted to study the suppression activity of the proinflammatory cytokines and mediators as a characteristic of antiinflammatory action, by using stem extract of A. sessilis in the lipopolysaccharide- (LPS-) stimulated RAW 264.7 macrophage cell line. The results showed that the extract has significantly inhibited the production of the proinflammatory mediators including NO and PGE2; cytokines comprising IL-6, IL-1 $\beta$ , and TNF $\alpha$ ; and enzymes covering the iNOS and COX-2 by preventing the IkBa from being degraded, to inhibit the nuclear translocation of NFκB subunit p65 in order to hinder the inflammatory pathway activation. These results indicated that the stem extract of A. sessilis could be an effective candidate for ameliorating inflammatory-associated complications.